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DISCUSSION

Dr Ronald Fairman (*Philadelphia, Pa*). Based on where you are right now with this technology, what length uncovered stent do you currently favor when you're approaching a patient?

Dr Joseph V. Lombardi (*Camden, NJ*). Based on the data that we're seeing now, we're observing a lot of increased false lumen flow. I think the natural tendency of everyone is to increase your coverage zone down to the celiac artery for your proximal piece. However, the STABLE trial has demonstrated a very low 1.2% paraplegia rate and I think one of the advantages of using one short-body TX2 device followed by a long bare stent. Theoretically, the observed enhanced false lumen flow has minimized paraplegia and combined with low mortality rates, it seems to do the job while minimizing complications.

As far as the bare stent, from my standpoint, I think it comes down to what your patient looks like anatomically. If they have extensive malperfusion distally, then I would extend that bare stent all the way down to the bifurcation; if it looked like there was a very focal area, then I would minimize its coverage.

Dr Jon Matsumura (*Madison, Wisc*). What is the definition of thoracic versus abdominal aorta? Is it at the diaphragm? Is it at the celiac? Does abdominal aorta growth mean that these patients will require a type IV thoraco-abdominal repair?

If the abdominal aorta is defined at the celiac, have you modified your treatment in that when you need to go back and treat patients with abdominal aortic growth either with another renal stent or maybe an open repair, how do you feel about having the bare aortic stent there? Do you feel like there is a different approach you should do at that initial operation that might make that second repair simpler?

Dr Lombardi. We define the thoracic aorta from the celiac and above, abdominal is below, based on our core lab data, and that's how they measured our false lumen patency.

Now, if you have a bare metal stent crossing through the perivisceral aorta, which 80 of our patients had, and then you notice growth, having that stent there approximates the intima with your target visceral artery. So if you're looking to bring back the target vessel that has a reentry tear back in continuity with the true lumen, it's much easier with the bare stent present. Working through that stent is pretty facile, just like a fenestrated stent graft at the end of it all. There are smaller little intimal tears that are from the intercostals that may persist; however, when you tackle the large entry tears, you usually minimize flow to the degree where you can stabilize growth.

Dr Juan Parodi (*San Isidro, Argentina*). I have a question. It seems that we don't have solutions yet for the abdominal aorta in the long term, and we see these more and more as we follow our patients' dilatation of the abdominal aorta in type IIIb. It seems that with your system you can achieve positive remodeling of the true lumen. The problem is the false lumen. We are conducting studies in which we are seeing that the diastolic pressure in the false lumen is higher than in the true lumen and through numerical models, and, mechanical models and some anecdotal measurements, we are seeing that we need another treatment to complete this, either do percutaneous septotomy or perhaps using branches and complete the occlusion of the entry sites of the distal aorta. What do you think about it?

Dr Lombardi. I believe that's largely true. Persistent flow in the false lumen and the abdominal aorta is the Achilles' heel and responsible for growth. I think from a basic standpoint covering the reentry tears and minimizing flow into that segment has substantial value in the setting of growth. It's more or less anecdotal at this point because we don't have a host of patients to really to compare with those who did not achieve those procedures. Secondary procedures, particularly if patients are treated in the acute phase, are going to be common to this pathology and management.

Dr Manju Kalra (*Rochester, Minn*). I just have two quick questions for you. Do you have a theoretical explanation for why you had continued growth in the group of patients that were treated in the acute phase within the first 2 weeks compared to those treated later? And do you think, when you have a larger group of patients, you're going to be able to make a recommendation as to preferred timing of treatment based on this data?

Dr Lombardi. That's the question, isn't it? From the standpoint of patients with malperfusion and rupture, you have no choice. So you're treating them when they present. Most of the patients who present in the acute phase have those two problems. So from a standpoint of watchful waiting, and potentially waiting after 14 days to treat them, that might provide you some value in patients in whom you think you can wait on, but again that clinical scenario rarely will come up.

As to why it happens, I don't know. I think that in the acute phase there is inflammation and a very suggestible intimal membrane which maybe you set off a course or a series of events where the aorta just is unable to stabilize. But I don't have a very good explanation for that at this time. We clearly need to follow those patients closely, however.

Dr B. Timothy Baxter (*Omaha, Neb*). It looked like your rupture rate was higher in the growth group versus the no-growth group. Where are the ruptures occurring in these patients, and is there something that you could be looking for to try to prevent the rupture?

Dr Lombardi. We were unable to get an autopsy on the patients who ruptured, so we really don't know exactly where that occurred anatomically. They presented with back pain and most of them expired before we could fully evaluate the location of their ruptures. But we suspect they were all in the thoracic aorta.

However, you mentioned the increased rate of rupture in patients in the thoracic aorta with growth; although not significant, we had an equally converse situation in the abdominal aorta where the patients who had no growth had a higher mortality and rupture. So that was a little counterintuitive, not something I would have predicted; however, that's how it worked out. So, based on the data thus far, we can't predict rupture based on morphology and growth.